Variations of Left Ventricular End-Diastolic Pressure, Volume, and Ejection Fraction with Changes in Outflow Resistance in Anesthetized Intact Dogs

By Anastasios G. Tsakiris, M.D., Russell A. Vandenberg, M.B., Natalio Banchero, M.D., Ralph E. Sturm, and Earl H. Wood, M.D., Ph.D.

ABSTRACT

Multiple measurements of left ventricular volumes, ejection fractions, and circulatory pressures were made under conditions of varied outflow resistance in five intact anesthetized dogs by using a biplane videoangiographic recording system which displays simultaneously two roentgen images on the same television screen, records the images on video tape, and requires low radiation levels and small amounts of contrast medium. Aortic pressure was controlled by intra-aortic infusion of angiotensin or acetylcholine. With spontaneous sinus rhythm and normal aortic pressure, end-diastolic volume averaged 35 ml (2.3 ml/kg), and ejection fraction averaged 47% of end-diastolic volume. When aortic pressure was increased, average end-diastolic volumes increased to 53 ml and ejection fractions decreased to 29%. Conversely, when aortic pressure was decreased, average ejection fraction increased to 73%, although changes in stroke volume were usually not large under these conditions. Concomitant large changes in the shape of the left ventricular cavity were also observed. Similar changes were observed when heart rate was maintained constant by electrical pacing of the atria and ventricles with a constant atrial-ventricular stimulus interval. The variability in left ventricular volume and ejection fraction observed in the same animal during different circulatory states may account in part for the differences in values for these variables reported by different investigators.

ADDITIONAL KEY WORDS

changes in aortic pressure
acetylcholine angiotensin
biplane videoangiography
vasodilatation vasoconstriction
stroke volume
geometric form of the left ventricle in the same intact anesthetized dogs during induced changes in aortic pressure. To eliminate the effect of changes in heart rate on left ventricular volume, observations were carried out in the same animals under constant heart rates as well as during spontaneous sinus rhythm.

Methods

MEASUREMENT OF LEFT VENTRICULAR VOLUME

A detailed technical description of the video system is being published elsewhere (8). In this system the fluoroscopic images from two orthicon intensifier assemblies, positioned at right angle to one another and aligned in the same vertical plane, are displayed simultaneously on the same television screen. Because the output of each image-intensifier tube is scanned from left to right, the image from the vertically positioned x-ray tube is displayed in the left half of the video field and the one from the horizontal tube, in the right half. A special switching circuit is used to display the video picture from each system in a single video field at a field repetition rate of 60/sec. These video signals are recorded on magnetic tape which has an accessory data channel for simultaneous recording of the electrocardiogram or a pressure pulse, the movement of the injection syringe plunger, and a binary decimal identification code. Thus, the synchronization problems in the analysis of single frames become minimal.

Because of the sensitivity of the intensifier assemblies, use of small amounts of contrast medium (69% Renovist, 0.2 to 0.4 ml/kg body weight as compared to 0.8 to 1.2 ml/kg in conventional cineangiography) and low radiation (1 ma) produces a well-defined contour of the left ventricle and allows multiple injections in the course of an experiment. Recordings of left ventricular pressures during injections of the contrast medium revealed no or small increases of systolic pressure (2 to 6 mm Hg) and of end-diastolic pressure (0 to 1 cm H2O) (Fig. 1). Extrasystoles during the injection were uncommon when the position of the left ventricular catheter was adjusted so that its tip did not impinge on the wall of the ventricle. A 35-mm camera, focused on the face of the television monitor with its shutter synchronized with the electrocardiogram recorded on the accessory data track of the video tape, was used to obtain single-frame pictures at end-diastole and end-systole with exposure times of 1/60 second.

The ventricular images were projected on graph paper by using a film reader, and tracings were made of the ventricular boundaries. Calculation of ventricular volume was made according to the method proposed by Chapman and associates (6). The diameters of the left ventricular silhouettes were measured at 1-mm intervals over the full extent of each projection. The volume of each cross section of the ventricle was then calculated, assuming an elliptical shape, by the formula:

$$V = \frac{\pi h (D_1 \cdot D_2)}{4},$$

in which $V =$ volume (ml); $h =$ height (0.1 cm); $D_1 =$ diameter of right anterior oblique projection (cm); and $D_2 =$ diameter of left anterior oblique projection at the same level (cm). The volumes of the 35 to 75 cross sections comprising the ventricle were then summed to obtain the volume of the ventricle. Corrections for geometric distortion were made on the basis of the roentgen screen-to-heart and screen-to-source distances.

![Figure 1](https://example.com/figure1.png)

**FIGURE 1**

Aortic and left ventricular diastolic pressures before, during, and after injection of 4.4 ml of 69% Renovist into left ventricle of a dog weighing 15.5 kg (morphine-pentobarbital anesthesia). Note absence of appreciable effect on systolic and diastolic pressures.
VARIABILITY OF LEFT VENTRICULAR VOLUME

PROCEDURE

Experiments were performed on five dogs weighing 14.0 to 16.5 kg. Each dog was premedicated with morphine, 2.5 mg/kg, and, 15 to 30 minutes later, anesthetized with sodium pentobarbital, 15 mg/kg, iv. A flexible, cuffed tube was inserted into the trachea, and intermittent positive-pressure (8 to 10 cm H2O) respiration with room air at approximately 30 cycles/min was used throughout the experiments. Respiration was temporarily suspended by opening the airway to ambient pressure during the periods of videoangiographic and pressure-pulse recordings.

Two bipolar electrode-catheters (6-F), introduced percutaneously via the left external jugular vein, were positioned so that their tips were in the right atrium near the superior caval orifice and in the outflow tract of the right ventricle, respectively. They were connected to coupled pacemakers so that the heart rate could be maintained constant by electrical stimuli at a constant atrial-ventricular stimulus interval. A third 8-F catheter was introduced via the same vein, and its tip was positioned in the pulmonary artery for the injections of indocyanine green. Two 5-F catheters were introduced into the femoral arteries and advanced into the thoracic aorta and the aortic arch just proximal to the origin of the brachiocephalic artery; the first was used for pressure recording and the second, for the infusion of drugs. Indicator dilution curves, for the determination of cardiac output, were recorded via a densitometer connected to a 20-cm nylon catheter whose tip was positioned in the abdominal aorta via a 17-gauge needle in the left femoral artery.

In four dogs, a Statham catheter tip manometer (model SF1) was advanced from the right carotid artery into the left ventricle for continuous pressure recording. In these animals, contrast medium was injected into the left ventricle via a 100-cm 7-F, blind-end, spray-tip (Rodriguez-Alvarez) catheter positioned in the left ventricle by transseptal puncture from the right external jugular vein. In the fifth dog, this catheter was used for both pressure recording and injection of contrast medium. A high-sensitivity recording of the diastolic phase of the left ventricular pressure pulse was obtained by bounding the output of the strain gauge by means of an analog computer so that the recorded pressure was clipped at 40 cm H2O. Pressures were measured by Statham P 23Dd or P 23G strain gauges and recorded photographically and on magnetic tape. The animals were placed in a left lateral half-body cast1 which was tilted and fixed in position on the fluoroscopic table so that oblique projections were obtained on the two image-intensifier screens. The injections of the contrast medium were synchronized with the cardiac cycle by an electronically controlled syringe activated by the R wave of the electrocardiogram and usually were completed during the diastolic phase of the cardiac cycle.

Intracardiac and intravascular pressures, cardiac output, and left ventricular volume at end-diastole and end-systole were measured in the "control state" and during induced changes in aortic pressure. All measurements reported herein were made under steady-state conditions attained 3 or more minutes after induced changes in aortic pressure, when the circulatory pressures had remained constant for 2 or more minutes.

Aortic pressure was modified by the continuous infusion of angiotensin II, 0.10 to 0.25 μg/kg/min, or acetylcholine chloride, 5.44 to 12 μg/kg/min, into the ascending aorta, 3 to 4 cm above the origin of the coronary arteries. When infused at this site, both pharmacologic agents had mainly a peripheral vasoactive effect, circulatory pressures and blood flow returning promptly to control levels when the infusion was stopped. In the intact animal, acetylcholine administered in this manner produced a peripheral vasodilatation with increased venous return and marked increase in cardiac output (up to two- or three-fold above control values), a moderate decrease in aortic pressure, and a reflexogenic increase in heart rate. These circulatory changes are somewhat similar to those associated with exercise (9). To eliminate the effect, on left ventricular volume, of the reflex changes in heart rate associated with the induced changes in aortic pressure, observations were made in each animal during spontaneous sinus rhythm and while the atria and ventricles were being driven at constant rates of 161 to 174 beats/min, with an atrial-ventricular stimulus interval of 80 to 90 msec.

Results

Results of the studies during spontaneous sinus rhythm are summarized in Table 1; those obtained at constant heart rates are shown in Table 2.

OBSERVATIONS DURING SPONTANEOUS SINUS RHYTHM

The average end-diastolic volume for the five dogs in the "control state" was 35 ml (range, 29 to 45) and the average ejection fraction was 47% of the end-diastolic volume (range, 39 to 53). The increase in mean

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1Supplied through the courtesy of David Clark and Co., Worcester, Mass.
### Changes in Circulatory Variables during Induced Changes in Aortic Pressure in Dogs under Morphine-Pentobarbital Anesthesia

<table>
<thead>
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<th>Dog</th>
<th>Wt. (kg)</th>
<th>Experimental condition</th>
<th>Drug dose (μg/kg/min)</th>
<th>Heart rate (beats/min)</th>
<th>Mean aortic pres. (mm Hg)</th>
<th>Stroke vol (ml)</th>
<th>LVEDP* (cm H₂O)</th>
<th>EDV† (ml)</th>
<th>Ejec. fract. (% EDV)</th>
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* LVEDP* = left ventricular end-diastolic pressure; zero reference point: midchest level.
† EDV† = left ventricular end-diastolic volume.
TABLE 2

Changes in Circulatory Variables during Induced Changes in Aortic Pressure with Constant Heart Rate in Dogs under Morphine-Pentobarbital Anesthesia*

<table>
<thead>
<tr>
<th>Dog</th>
<th>Heart rate (beats/min)</th>
<th>Mean aortic press. (mm Hg)</th>
<th>Stroke vol (ml)</th>
<th>LVEDP (cm H2O)</th>
<th>EDV (ml)</th>
<th>Eject. frac. (% EDV)</th>
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*Dogs, symbols, and other experimental conditions identical to those in Table 1.

FIGURE 2

Left ventricular end-diastolic volume and ejection fraction before, during, and after changes in aortic pressure induced by intra-aortic infusion of angiotensin and acetylcholine in a dog (weighing 14.8 kg) under morphine-pentobarbital anesthesia. ED and ES show single video fields from biplane left ventricular angiograms with their 1/60-second exposure times electrically synchronized to occur at end-diastole and end-systole, respectively. Left picture of each pair is anterior oblique projection obtained by vertically oriented video roentgenographic system. Note large variations in volume, form, and ejection fraction of left ventricle associated with relatively small changes in stroke volume.
aortic pressure during administration of angiotensin (average increase, 48 mm Hg) resulted in an increase of end-diastolic and residual volumes in all instances and, consequently, in a considerable decrease in the ejection fraction (average, to 29%) while stroke volume remained practically unchanged. The increase in end-diastolic volume (average, 50%) was associated with increased end-diastolic pressure and longer duration of systolic ejection.

The opposite changes were observed when the resistance to left ventricular outflow was reduced by infusion of acetylcholine. The average decrease in mean aortic pressure was 17 mm Hg. The slightly greater stroke volume (average increase, 15%) was ejected from a smaller end-diastolic volume (average decrease, 22%) while the average ejection fraction was increased to 73% (range, 66 to 80) of the end-diastolic volume. The left ventricular cavity at end-systole was very small (Fig. 2). The average decrease in left ventricular end-diastolic pressure was 5 cm H2O.

Figure 3 displays a representative example of changes in end-diastolic volume and pertinent hemodynamic parameters when the heart rate was increased from 101 to 200 beats/min by pacing the atria and ventricles at a constant atrial-ventricular stimulus interval of 80 msec. A progressive decrease in end-diastolic volume was associated with an increase in end-diastolic pressure when the heart rate was increased above levels of 135 to 165 beats/min.

OBSERVATIONS AT CONSTANT HEART RATE

When the heart was driven at a rate of 161 to 174 beats/min, the end-diastolic volumes (average, 28 ml), ejection fractions (average, 37%), and stroke volumes (average, 10 ml) were smaller in all five dogs than when the heart was allowed to beat at its spontaneous sinus rate of 84 to 104 (average, 96) beats/min (Tables 1 and 2).

When the heart rate was maintained constant during infusion of angiotensin or acetylcholine, the changes in ventricular volume and ejection fraction were directionally similar although smaller in magnitude than the
Left ventricular end-diastolic volume and ejection fraction before, during, and after changes in aortic pressure induced by intra-aortic infusion of angiotensin or acetylcholine. Heart rate and temporal relationships of atrial and ventricular systoles were maintained constant by electrical pacing. Deflections marked A and V in ECG tracing are produced by the electrical stimuli driving the atria and ventricles, respectively. Variations in ventricular volume, ejection fraction, and end-diastolic pressure were similar to those obtained with spontaneous sinus rhythm in this same dog (Fig. 2).

Changes observed during sinus rhythm (Tables 1 and 2). With angiotensin, end-diastolic volume increased an average of 39% above the control values and ejection fraction decreased to 24% with practically no change in stroke volume. After acetylcholine, decreased aortic pressure (average decrease, 17 mm Hg mean pressure) was associated with a decrease in end-diastolic volume in three dogs and no change in two, while stroke volume and ejection fraction increased. The average ejection fraction (68% of end-diastolic volume) during acetylcholine infusion was only slightly lower than that observed with spontaneous sinus rhythm. The left ventricular videoangiograms showed that at end-systole the cavity was extremely small and the apical region was practically obliterated (Fig. 4).

End-diastolic pressure varied directly with end-diastolic volume; however, at these relatively fast, constant heart rates, given levels of end-diastolic pressure were associated with smaller ventricular volumes than those observed at the slower sinus rhythm rates. This change in pressure-volume relationship was studied in detail in an additional dog (Fig. 5) and is probably due, at least in part, to the inadequate time for ventricular relaxation during diastole (10) at the relatively fast heart rates required to maintain control of the heart under these circumstances.

Discussion

The volumes of the left ventricular chamber during the various phases of the cardiac cycle are among the most important determinants of cardiac function. However, relatively little information is available concerning successive determinations of the internal volume and shape of the left ventricle under different hemodynamic conditions in the same intact animal. The data available so far have been mainly collected by using indirect methods (1,3,5).

The videoangiographic system used for the
Current study permits repeated direct measurements of left ventricular volume at 1/60-second intervals in the same animal. We think that, because only small amounts of contrast medium are used for each videoangiogram, the effects of the injected material are minimized (11). In the present experiments, changes in aortic pressure, induced by intra-aortic infusion of vasoactive drugs (so that direct cardiac effects were minimal), were used to study the variations in left ventricular volume under different controllable hemodynamic conditions in the same animal. The induced changes in aortic pressure were associated with reflex changes in autonomic tone and consequently in heart rate. An effort to minimize the chronotropic effects of the variations in autonomic tone was made by maintaining the heart rate constant by external pacing of the atria and ventricles. However, the constant rates were relatively fast (161 to 174 beats/min) because it was necessary to exceed the spontaneous rates encountered during intra-aortic infusion of acetylcholine.

The directional and relative changes in left ventricular volume and end-diastolic pressure associated with increases or decreases in aortic pressure were similar during observations with spontaneous and with driven rates. The dramatic changes in end-diastolic volume and ejection fraction observed under these conditions are partly due to the induced changes in sympathetic and parasympathetic outflow to the heart as evidenced from studies in dogs with chronic or acute cardiac denervation (12).

Review of individual frames at end-diastole and end-systole showed that, in the control state and when aortic pressure was increased, ventricular ejection was primarily accomplished by shortening of the transverse diameter of the chamber with relatively little...
shortening of the longitudinal axis (distance between aortic valve and apex). The shortening of the long axis under these conditions did not exceed 10% (range, 4 to 9.5) of the end-diastolic length. With acetylcholine, however, in addition to the reduction of the transverse diameter, considerable shortening of the long axis of the cavity, varying between 10 and 25%, was observed. This resulted in a more complete emptying of the left ventricular cavity, especially in the apical region.

It is probable that essentially complete obliteration of the cavity occurs toward the apex so that it is not seen on the roentgenogram, resulting in overestimation of the actual degree of shortening of the longitudinal muscle fibers of the heart.

End-diastolic pressure changes in these studies were uniformly associated with similar directional changes in end-diastolic volume. The only instances of opposing changes in pressure and volume were observed when the heart was driven at rates greater than 145 to 165 beats/min, a circumstance in which very probably, in the absence of high levels of sympathetic tone, diastole is restricted to a period less than that required for full relaxation of the ventricle (10). It appears, therefore, that acute changes in end-diastolic pressure in the same heart will be associated with directionally similar changes in ventricular volume. The results obtained in anesthetized intact animals, under the controlled conditions of these experiments, cannot be extrapolated with acceptable certainty to the unanesthetized animal or man. They do show, however, the high degree of variability which can be expected in parameters such as end-diastolic volume, ejection fraction, and internal form of the left ventricular cavity during different types of circulatory stress in the same intact animal with a normal cardiovascular system.

Except when values are very low, a single determination of the ejection fraction does not appear to provide a satisfactory index of ventricular contractility, and hence it is not a satisfactory parameter for distinguishing between normal and impaired myocardial function. In the absence of accurately controlled conditions, it is questionable that single determinations of all pertinent hemodynamic parameters, including measurement of phasic ventricular volumes and pressures, would suffice to give a reliable index of the reserve capacity of the heart and hence distinguish between a normal left ventricle and one with moderately impaired contractility due to myocardial disease.

The variability in left ventricular volume and ejection fraction observed under different conditions in the same animal in these studies is probably responsible in part for the different values reported for these parameters by different investigators using different techniques and non-uniform hemodynamic conditions. Furthermore, a consideration of the variability in the above-mentioned parameters in the same ventricle is necessary when geometric models for mechanical analysis of ventricular contraction are constructed.

Acknowledgments

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References


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